COMMENTARY PEER REVIEWED

Evident similarity of porcine postparturient dysgalactia to subclinical porcine coliform mastitis

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Summary

The present commentary aims to motivate future research and initiate new investigation on porcine periparturient disorders. After a short characterization of the clinical presentation of coliform mastitis, this commentary concentrates on the subclinical variant. The subclinical form of the disease resembles in most aspects what is referred to as postparturient dysgalactia syndrome of sows, and is considered highly prevalent in the field. Since the recent introduction of the ill-defined postparturient dysgalactia syndrome, experimental work has declined. Except for review articles, there is a shortage of recent publications in this area. Previously published experimental data led to a promising approach to prevent coliform mastitis by reducing the level of teat contamination by coliform bacteria. With the ongoing need to reduce antimicrobial use in food-producing animals, there is a continued need to investigate preventive strategies.

Keywords: swine, review, mastitis, dysgalactia, prevention

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Resumen – Semejanza evidente entre la disgalactia porcina postparto y la mastitis coliforme subclínica

Este comentario pretende motivar estudios futuros e iniciar nuevas investigaciones sobre los trastornos periparto porcinos. Después de una corta caracterización de la presentación clínica de la mastitis coliforme, este comentario se concentra en la variante subclínica. La forma subclínica de esta enfermedad se parece en casi todos los aspectos, a lo que se llama síndrome disgalactico postparto de la cerda y se considera altamente prevalente en el campo. Desde la reciente introducción del mal llamado síndrome disgalactico postparto, el trabajo experimental ha declinado. A excepción de los artículos de análisis, existe una escasez de publicaciones recientes en esta área. La información experimental publicada anteriormente llevó a una estrategia prometedora para prevenir la mastitis coliforme al reducir el nivel de contaminación de la teta con bacterias coliformes. Con la necesidad actual de reducir el uso de los antimicrobianos en animales para consumo, existe una necesidad constante de investigar estrategias de prevención.

Résumé – Similarité évidente de la dysgalactie post-partum porcine et de la mammite subclinique à coliforme porcine

Le présent commentaire vise à motiver des recherches futures et à initier de nouvelles études sur les désordres péri-partum porcins. Après une brève caractérisation de la présentation clinique de la mammite à coliforme, ce commentaire se concentrera sur la variante subclinique. La forme subclinique de la maladie ressemble en plusieurs points à ce qui est appelé le syndrome de dysgalactie postpartum des truies et est considéré comme très prévalent sur le terrain. Depuis la récente introduction du syndrome mal défini de dysgalactie postpartum, le travail expérimental a diminué. À l'exception de quelques article de revue, il y a une pénurie de publications récentes sur le sujet. Des données expérimentales déjà publiées avaient mené à une approche prometteuse pour prévenir la mammite à coliforme en réduisant le niveau de contamination du trayon par les bactéries coliformes. Avec le besoin en cours de réduire l'utilisation d'antimicrobiens chez les animaux de rente, il y a un besoin continu à étudier des stratégies de prévention.

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During the sow's peripartum period, several disorders are frequently observed and of great significance to economics, animal welfare, and pressures to reduce antimicrobial use for prevention and therapy in food-producing animals. Reviews and textbook chapters published during the last four decades illustrate the diverse nomenclature used for these disorders and the absence of a generally accepted theoretical model of pathogenesis.[1-5] In the authors' view, the most recent nomenclature used for these disorders, postparturient dysgalactia syndrome (PPDS), does not account for knowledge achieved in earlier studies about the syndrome and we propose the term PPDS be revisited. The focus of the present commentary is on the gaps in the literature on previous experimental work and is meant to challenge and motivate researchers to revisit the syndrome and address these gaps with ongoing research.

Coliform mastitis

Brief characterization of clinical coliform mastitis

Coliform mastitis (CM) is a febrile peripartum disease, formerly called milk fever, most often observed during the first 24 h after parturition but can also be observed on the day before and up to 2 days post parturition.[6,7] In addition to pyrexia, clinical signs reported include reluctance to allow nursing, anorexia, constipation, thickened white vaginal discharge, increased respiratory rate, reluctance

to rise, signs of insufficient caloric intake in piglets, and lower weight gain in piglets.[2,6,8] These signs are nonspecific and not pathognomonic.[9] To a producer monitoring their animals closely, these clinical signs are more obvious than lesions in the udder.[10] Mastitis is caused by coliform bacteria, ie, members of the family Enterobacteriaceae, that invade through the teat duct.[11] The mode of the bacterial invasion and their spread within the mammary gland determine the irregular expansion of the inflammation.[12,13]

Diagnosis of CM

Classical signs of inflammation, such as swelling, firmness, increased local skin temperature, soreness, and reddening of the skin, may or may not be present. In one field study, only half of the mastitic glands were diagnosed by clinical examination where isolation of *Escherichia coli* was possible and where cytologic smears showed inflammatory cells.[14] In most cases, the udder lesions are limited to single complexes, subcomplexes, or foci of a few centimeters or less in diameter.[15] According to a comprehensive histological study based on 25 tissue blocks per inoculated gland, the most numerous foci and the most severely affected areas are situated dorsally in the udder, ie, close to the abdominal wall and therefore not easily accessible by manual palpation.[13] The glandular tissue is infiltrated by edema, fat, and covered by relatively thick skin.[2] During the critical time postpartum, a variable proportion of glands remain partially or totally unsuckled and therefore can remain congested with colostrum.[16]

These aspects of mastitis presentation in the sow support why it is often difficult to diagnose clinical mastitis even by careful external examination of the udder. This difficulty in diagnosis likely plays a role in the underdiagnosis of mastitis by many clinicians and producers with relation to post-parturient disorders.

A valid determination of mastitis and intramammary infection is only possible if milk samples are collected from each subcomplex of the gland.[2,8] If milked as usual, a sample from a teat is a composite sample of the secretions originating from the two subcomplexes. According to the experience of the authors, a mixed sample will primarily be composed of the secretion of the healthy or healthier subcomplex due to the higher viscosity of the secretion from the affected tissue. The low secretory activity of mastitis-affected foci will also decrease the ability to diagnose mastitis in samples from focally affected subcomplexes. The involution of complexes not suckled by a piglet renders the interpretation of cytological results difficult due to the increased total somatic cell counts (SCC) from such glands which may be significantly higher than counts from mastitic glands.[15,16] Markedly elevated counts of polymorphous neutrophils (PMN) are present as well in secretions from glands undergoing involution. Wegmann and Bertschinger[16] proposed the use of a threshold value composed of a combination of SCC and PMN to discriminate between involution and mastitis. Thus, a SCC of 5×10^6 cells/mL is indicative for mastitis if the proportion of PMN exceeds 70%.[16] The authors experience shows this threshold value is not applicable to samples obtained later than 2 days after parturition. In a more recent paper, a threshold for SCC of 2×10^6 cells/mL was used. However, Kotsarev et al[17] did not explain how they distinguished mastitis from involution without PMN determination.

To the authors' knowledge, a reliable and rapid test for on-farm diagnosis of mastitis is currently not available. Tests developed for use in cattle are not recommended due to the generally higher cell content of sow milk.[4] Additionally, the measurement of milk pH is of limited diagnostic value.[6,14]

A study comparing postmortem lesions in affected sows with control sows demonstrated a significant association between agalactia and mastitis.[6] However, necropsy results in mild cases were not necessarily reliable since inflammatory lesions may be too small to be sampled and affected areas cannot easily be differentiated from unaffected glandular areas macroscopically.[12] In view of the independent processes started by different organisms and at different times, this variability of the mastitic foci in a given sow is not unexpected.[2]

The sensitivity of histologic examination of affected glandular tissue is highly dependent on the way the udder is sectioned and on the number of tissue blocks examined. The most prevalent histologic findings in affected and control sows are edema and congestion.[6] In addition, affected areas show an acute catarrhal-purulent mastitis with conserved acinar structure of the gland. The lactic ducts are filled with epithelial and inflammatory cells. In more severe cases, extensive necrotic foci are sometimes surrounded by neutrophil demarcation.[12,13] Histological findings in other organ tissues have not been significantly correlated with the disorder.[9,12,15]

The results of bacteriological examination of tissue samples taken at the time of necropsy are more reliable than those based on milk samples, since the contamination with environmental or teat skin flora during milk collection is difficult to avoid. Samples from the teat skin and samples of milk exhibit a similar flora when analyzed following enrichment in a fluid medium.[18] This sampling contamination has led to variability and the confusing conclusion that secretions from glands with and without mastitis contain a similar bacterial flora.[5,19,20] Investigators who inoculated solid culture media directly with material from superficially sterilized affected mammary tissue could reliably identify Enterobacteriaceae.[21,22] Since inflammatory lesions persist for longer time periods than culturable bacteria, the latter cannot be consistently isolated from sites with microscopic lesions.[13,14,23] Other non-coliform bacteria, such as *Streptococci* and *Staphylococci*, are only rarely associated with mastitis of mild degrees.[22]

To improve on the effort to obtain a tentative diagnosis based on cytology and bacteriology of the secretion, there have been attempts to utilize clinical pathology criteria for the diagnosis of CM. A marked transitory leukopenia has been observed in experimental acute mastitis.[11,13] However, in field cases this finding is less marked, probably due to the variable time between the onset of an infection in individual glands and the sampling period.[15] Other investigators have observed and reported an increased erythrocyte sedimentation rate and a decreased ratio of plasma protein to fibrinogen.[8] These parameters were confirmed in sows with subclinical mastitis as well.[15] Following experimental inoculations, tumor necrosis factor-α and IL-6 were found to be promising markers for the severity of mastitis[24] while other acute phase proteins proved to be less specific.[24,25]

In summary, the diagnosis of CM in the field is still a challenge. Even with efforts to examine secretions from each gland, some degree of uncertainty in reaching a diagnosis remains.

Subclinical CM

Sows affected by CM may exhibit a wide range of clinical signs ranging from subclinical and

mild hypogalactia to severe mastitis with severe systemic signs.[26] The authors believe that subclinical mastitis, ie, mastitis without visible clinical signs, has not been thoroughly investigated and gaps in our knowledge remain. In postmortem studies comparing mammary glands from mastitis-affected sows to healthy control sows, a variable proportion of the latter showed inflammatory lesions in the glands as well. However, as a rule, foci were less numerous and showed a lower degree of inflammation.[9,12,15] It appears that spontaneous mastitis of all degrees of severity occurs more frequently than the severe cases would indicate. In a field study report, samples of colostrum from 59 sows from 15 herds with a mastitis problem were examined cytologically and bacteriologically. Eighty-three percent (49 of 59) of the sows were affected by mastitis and coliform bacteria were isolated from 71.2% (42 of 59) of the sows. However, no more than 39% (23 of 59) of these sows were febrile at the time of examination.[27] In a research swine herd with an extremely low incidence of clinical CM, subclinical mastitis diagnosed cytologically was detected in 61% (97 of 159) of the farrowings.[28] In a Swedish study based on a population of clinically healthy sows, mastitis with pure cultures of *E coli* and significantly increased SCC was observed in 15.6% (15 of 96) of farrowings on the first day of lactation.[14] Persson et al[14] reported that the bacteria were eliminated between days 3 and 8 of lactation. The importance of subclinical mastitis was also emphasized in a recent study by Kotsarev et al.[17] However, since the exclusion of glands undergoing involution was not mentioned and only SCC were reported, the results cannot be directly compared to other studies.

Additionally, the economic significance of subclinical CM has received little attention to date. The increased erythrocyte sedimentation rates and the decreased ratio of plasma protein to fibrinogen in sows with subclinical mastitis indicate a negative health effect of the disease in affected sows.[15]

Effect of subclinical CM on suckling piglets

Data on milk yield of sows with subclinical coliform mastitis are rare. When average daily gain of piglets from spontaneously agalactic sows with clinical CM was compared to piglets from unaffected sows, piglets from agalactic sows lost weight on the first 2 days postpartum and gained significantly less weight on the third day.[8,29] Sows experimentally inoculated with *E coli* experienced high piglet mortality due to piglet starvation. The surviving piglets grew significantly slower on days 1 through 3 of age but were not significantly lighter at 14 days of age than piglets from resistant and non-inoculated sows. The experimentally inoculated sows that did not develop mastitis had no piglet mortality compared to litters of susceptible sows and piglet average daily gain through 14 days of age was identical to control litters suckling non-inoculated sows.[30] Piglet weights and health of the mammary glands suckled by the piglets were sequentially recorded in a study focusing on the hygiene of the farrowing environment.[23] Subclinical CM developed in 16 of 24 sows (66.7%). Piglets suckled 64.9% (185 of 285) of the healthy glands and 43.5% (27 of 62) of the glands with positive cytology. During the first 4 days of lactation, piglets suckling healthy glands had an average daily gain of 125 g as compared to 105 g in piglets which had suckled glands with mastitis. Average daily gain from day 5 through day 21 was identical regardless of the gland suckled.[23]

In another project studying the influence of four farrowing systems on CM, 159 farrowings were observed. Clinical CM developed in 4 farrowings and subclinical CM was diagnosed in 97 farrowings. The percentage of piglets dying of starvation increased linearly to the incidence of mastitis.[28] Increased piglet mortality is most often caused by starvation. Piglet mortality in a litter is negatively correlated with the weight gain in the first 3 days of life of the surviving litter mates suggesting that low milk production by the sow is associated with piglet starvation.[31] Thompson and Fraser[32] saw marked variation in weight gain within litters in the first 3 days of life. Average daily gains of piglets were negatively correlated with the rectal temperature of the sow. Litters with low initial gains showed more variable gains as well. Such litters were not associated with obvious mastitis suggesting that subclinical disease of the sow might lead to inadequate milk production.[32] In a Swedish study based on 369 farrowings, piglet mortality in the first week and the within-litter standard deviations for weights at 3 weeks of age were correlated to the rectal temperature of the sow in the first 48 h post parturition. Many of these correlations were significant even though the sows affected by clinical mastitis were omitted from the analysis. This indicates that subclinical mastitis negatively affects production performance.[33]

Factors affecting CM severity

The virulence of the pathogen can normally be considered an important factor for the severity of the subsequent disease. However, in the case of CM, such an influence is not well documented. When sows are inoculated intramammarily, the course of the experimental disease induced with identical bacterial cultures may vary greatly.[13,26,30,34] The number of bacteria inoculated does not explain the variable outcome.[1,11,30] Outbreaks of the severe form of the disease have been reported in which almost all sows farrowing over a period of several weeks may be affected and then suddenly no further cases develop for no evident reason.[2] This observation serves as an argument against an increased susceptibility of certain sows. In accordance, sequential observation of 39 sows over six consecutive farrowings resulted in no evidence for individual disposition to CM.[7]

Sows from a specific-pathogen-free herd were resistant to a standardized experimental infection, whereas sows from a conventional herd were highly susceptible.[30] One of several explanations to support these divergent outcomes could be an inapparent viral or bacterial infection in the conventional herd leading to some sort of immunosuppression. Further, a functional difference was detected in the PMN of susceptible sows possibly indicating impaired PMN function.[35] This latter result differs from the findings from a study involving experimental inoculation of 12 sows shortly before parturition. Four sows developed clinical mastitis but this did not appear to impact the functional traits of the circulating granulocytes such as chemotaxis, phagocytosis, or CD18 expression. Österlundh et al[34] concluded that factors other than granulocyte function determine whether a sow will develop clinical mastitis following infection with *E coli*.

Bacteria in the secretion within the mammary gland are immediately exposed to a new microenvironment. Their proliferation is an important factor for the host-microbe balance. The severity of experimental CM depends on the proliferation of the inoculated bacteria. Numerical estimates of the bacteria sequentially recovered from the secretion indicate that glands of susceptible sows harbor substantially more organisms than resistant sows.[30] However, further information to support this finding is scarce. Two strains of *E coli* isolated from CM grew significantly faster in lactoserum taken on

the day of farrowing compared to lactoserum sampled later. These *E coli* strains also grew faster in lactoserum from sows affected with mastitis compared to lactoserum from healthy sows.[36] When a greater number of isolates were examined in untreated colostrum and milk, the situation appeared to be more complex. Not all isolates from porcine CM behave in a similar manner. There are isolates with much slower growth.[37] In addition, there is variation in the bacterial growth rate in secretions from individual sows.[37] In secretions from a healthy, suckled gland, a strain of *E coli* exhibited continual growth throughout lactation, whereas the viable count of the same strain remained either constant or was even reduced in the secretions from healthy, non-suckled glands or from glands with mastitis.[37] The latter finding corresponds with the spontaneous elimination of the organisms from infected glands within about one week.[37]

During gestation, the mammary secretion strongly reduces the growth of coliform bacteria. Following experimental intramammary inoculation two days prior to parturition or external contamination of the teat orifices at the same time, signs of mastitis develop only after the start of parturition.[11,26] The mechanism of this inhibition is not known.

Mammary gland exposure to coliform bacteria

Coliform mastitis is an example of a non-contagious infectious disease. Under the conditions of outdoor pig production, it is rare.[2,6] In 9 Danish herds with outdoor farrowing systems, only 1.1% (13 of 1206) of sows developed CM as compared to 17.1% (286 of 1674) of sows from 9 other herds managed in a traditional indoor confinement system.[38] These observations may indicate a fecal contamination of the teats as a potential source of the infection.

To further test the role of fecal contamination of the teats, 12 sows farrowed in an experimental pen designed to allow the sow to choose where to lay and 12 sows farrowed in a conventional farrowing crate. Viable Enterobacteriaceae counts were performed from the floor in the laying area and from the surface of every teat apex from 3 days before until 1 day after parturition. Colostrum was collected from every teat beginning immediately after parturition and repeated every 12 h. Bacterial counts on the floor and the teats differed between the two systems by a factor of 10 to 1000. Furthermore, *E coli* was isolated from 3 mammary glands in the experimental pen as compared to 27 glands in the conventional crate and about half of the infections were detected in the first sample collected after parturition. In glands with positive cytology but no viable bacteria, mastitis must have been of shorter duration.[23]

Farrowing systems were compared under field-like conditions in another study.[28] Forty sows were assigned to one of four farrowing systems. Viable counts of coliforms on the teat ends were done on gestation day 112 and colostrum was aseptically collected once within 36 h after parturition. Clinical CM was very rare, but subclinical mastitis developed in 61% (97 of 159) of the farrowings. The incidence of mastitis was significantly dependent on the design of the farrowing system which differed with regard to the separation of the areas for laying and for defecation. The incidence of mastitis correlated to enterobacterial counts on the teat ends.[28]

Prevention of CM

Research on CM has identified ways to prevent the disease by protecting the teats from contamination with coliform bacteria during late gestation and the first 3 days of lactation. Coliform bacteria are natural inhabitants of the digestive and urinary tract of the sow. The farrowing system should be designed in a way to prevent the sow from lying in her own excreta. Visual absence of any fecal traces on the ventral skin of the sow is a simple criterion, but not always easy to accomplish with indoor climatic conditions changing throughout the year. If coliform bacteria can successfully be kept away from the lactiferous system of the gland, other prophylactic measures, eg, antibiotic treatment, become less necessary.[23,28]

The role of the sow's own microbiome as a reservoir of coliform bacteria sheds light on the reasons surrounding failure of sanitation measures for the prevention of CM.[2,6] For example, details such as the type of bedding may be important. In a survey of 3000 farrowings in units where wood shavings were used as bedding, 180 (6%) of the farrowings needed treatment for mastitis. Following a change to straw bedding, the incidence over the next 1800 farrowings dropped to 2.5% (45 farrowings).[39] In cattle, where coliform mastitis causes significant loss, maintenance of low levels of coliform bacteria in the bedding is the only effective method of control.[40] Coliform bacteria have the capacity to pass through the bovine streak canal between milking times. The frequency of this event depends on the number of organisms applied.[40] Highest coliform counts are found in sawdust bedding. Incubation of contaminated sawdust at temperatures above 22°C has been reported to allow 1 or 2 $\log_{10}$ proliferation of the organisms.[40]

Postparturient dysgalactia syndrome

Terminology

Many clinically healthy sows nurse litters with increased mortality and poor and uneven growth rates. The various physiological processes occurring around parturition make it difficult to differentiate health problems in these apparently non-diseased sows. Therefore, the early lactation problems should be described as PPDS. That term is preferred over the more traditionally used mastitis-metritis-agalactia (MMA) syndrome.[41] Variations in criteria, assessments, and reporting explain the difficulty to precisely define PPDS. A differentiation of PPDS from MMA is not clearly possible.[5] Careful investigations revealed that metritis is rather uncommon in sows with problem litters[10] and complete agalactia is a very rare exception. Thus, the authors believe that MMA has become a widely used misnomer. Reiner et al[42] preferred the term PPDS over MMA because lactational failure can be a consequence of different pathological processes and lactational failure is the cardinal sign of the economically important average daily gain of piglets. Coliform mastitis is considered a subtype of PPDS[43] or the emerging tip of the iceberg represented by PPDS.[20]

Diagnosis of PPDS

Reports on clinical trials in PPDS are quite rare. Epidemiological research often relies on data collected by animal caretakers. Not all signs must be expressed at the same time in the same sow so a generally accepted clinical description for PPDS based on objective parameters does not exist.[44] Increased rectal temperature was nearly always mentioned as a clinical sign.[19,25,44-47] However, reference values for body temperature are inconsistent with recent findings in healthy sows.[48,49] Other clinical signs recorded include external signs of mastitis,[19,25,44-47] anorexia,[19,25,46] appetite measured indirectly as

change in backfat thickness through lactation[45,50] or body weight,[50] constipation, or vaginal discharge.[25,45,46,50]

Piglet vitality has been assessed by recording mortality during the first weeks of lactation,[45,50] piglet daily body weight gain[45,50] or observation of changes in piglet behavior.[19,44,47] A weakness of the literature is that some investigations into piglet nutrition and development were performed without a detailed look at the health of the sow.[51-53] Colostrum yield through 24 h postpartum was calculated from the weight change of the litters.[51] The wide variability of colostrum yield was attributed to hormonal, environmental, and nutritional factors. Surprisingly, litter size is known to have a strong influence on milk production but does not affect colostrum yield.[51] Sows producing a low amount of colostrum were characterized by leaky mammary epithelium and reduced synthesis of lactose, which may be related to hormonal changes prior to parturition.[52] The within-litter variation of piglet performance until 3 weeks of age appears to be significantly correlated with birth weight and milk intake, whereas birth order and location of the preferred teat do not have a significant influence.[53] A recent review emphasizes the need for more research to improve the yield and composition of colostrum, yet makes no reference to sow health.[54] In summary the authors feel that veterinarians, epidemiologists, and animal science researchers have investigated similar shortcomings of piglet performance without drawing on the expertise of supporting professions.

Etiology and pathogenesis of PPDS

A primary difficulty is the establishment of a functional definition for PPDS. Microbiologists see PPDS primarily as an infectious disease, endocrinologists see it as a hormonal disturbance, and others consider it a nutritional disease.[55] Several studies attribute a central role to endotoxins.[42,43,55] It is well established that inoculation of endotoxin into a pig's circulation induces a systemic disorder which mimics the general signs of CM, whereas oral administration of endotoxin does not induce obvious clinical symptoms. The pig is the least sensitive of mammalian species to parenterally applied endotoxin.[56] Furthermore, local mammary lesions have never been reproduced by endotoxin application except when given intramammarily and endotoxin has been detected in the blood of affected sows only in a minority of CM cases.[57] In a manner similar to that seen in ruminants, De Ruijter et al[58] showed that coliform mastitis-causing bacteria in sows induce acute-phase mediators, which are locally released into the mammary gland, enter the circulation, and act on other tissues including the thermoregulatory central nervous center. Endotoxins, however, essentially remain in the affected gland. Extremely high doses of endotoxin must be inoculated into the mammary gland to be detectable in the blood.

A team of experts in PPDS have presented a new explanation for the syndrome calling it a change in homeorhesis, ie, a fault in the orchestrated changes in metabolism of body tissues necessary to support a physiological state like gestation or lactation.[20] They propose the dys-homeorhesis occurs during the shift from gestation to lactation inciting the development of PPDS and that the physiopathology of PPDS includes feed and feeding in gestation, endotoxemia, and stress. The concept of dys-homeorhesis is broadly discussed at length but the intended benefit of the new theory remains unclear.[20]

Reports addressing results of laboratory examinations of mammary secretions or of necropsy results of sows affected with PPDS were not identified by the authors. Nor has detailed literature regarding the performance of litters from affected sows been detected. The implied multifactorial nature of PPDS[20,42,43] may limit investigators from analyzing the syndrome in more detail. It must be kept in mind that any factor affecting the lactation performance of the sow is defined to take part in the syndrome. In consequence, the search for disease causing or disease accelerating mechanisms may be unrewarding. The authors believe the introduction of PPDS as a new concept coupled with the perceived decrease of active research in CM is very likely due to the idea that the new concept would offer alternative solutions to the problem.

The search for risk factors for PPDS is the only research field where some publications have appeared. The risk factors are typically linked with multifactorial diseases. Many of the factors were found to have minor effects if present independently but found to cause disease if more than one were present.[5] From field observations, the associations of risk factors are not additive but synergistic.[55] Risk factors are often identified based on questionnaires completed by herd owners who diagnose sows affected by PPDS. Examples of statistically significant risk factors are feed and feeding regime, housing, management practices,[43] time of moving sows to the farrowing unit, farrowing induction, feeding sows ad libitum during lactation, frequent farrowing supervision,[59] integration of gilts into the herd after the first farrowing, firm fecal consistency in gestating sows, soiled troughs in lactating sows, low water flow rate in drinking nipples, and high prevalence of lameness.[60]

Similarities between PPDS and subclinical CM

The authors propose that PPDS presents with evident similarities to subclinical CM. The characteristic period of occurrence immediately before and after parturition, the clinical signs in the sow such as fever, anorexia, reluctance to nurse and move, increased vaginal discharge, reduced milk production in the absence of gross mammary lesions, and insufficient milk supply for piglets do not allow distinction of the two affections (Table 1). Clinical CM is considered the proverbial tip of the iceberg of subclinical CM as well as for PPDS.[20] Both PPDS and subclinical CM occur at a high incidence and are assumed to be the cause of uneven development of piglets and litters. Differences are restricted to limited laboratory results for mammary secretions and necropsies in the case of PPDS as well as speculative explanations about the etiology and pathogenesis of PPDS.[55]

Implications

- In view of the wide distribution of subclinical CM and PPDS, investigations into the potential relationship between these two conditions remain an important research area.
- Should further research support that the syndromes are indistinguishable from each other, the development of economically sustainable farrowing systems that aim to reduce the incidence of the syndromes is necessary.
- So far, PPDS is only described in the porcine species and so it is the opinion of the authors that PPDS is indistinguishable from subclinical CM and that a return to the use of subclinical CM terminology will allow future investigators to take advantage of the many parallels to CM in the bovine species and thus inspire new research approaches.

Table 1: Studies reporting clinical signs observed in sows diagnosed with subclinical coliform mastitis or postparturient dysgalactia syndrome

Clinical sign	Published reference	
	Subclinical coliform mastitis	Postparturient dysgalactia syndrome
Fever	Blood et al[2] Martin et al[6] Persson et al[7] Hermansson et al[10] Ross et al[15]	Pendl et al[25] Preissler et al[44] Claeyé et al[45] Tummaruk and Sang-Gasanee[46] Guillou et al[50]
Anorexia	Blood et al[2] Persson et al[7] Hermansson et al[10]	Pendl et al[25] Tummaruk and Sang-Gasanee[46]
Reluctance to rise	Blood et al[2] Martin et al[6] Hermansson et al[10]	Pendl et al[25]
Constipation	Blood et al[2] Martin et al[6] Hermansson et al[10]	Pendl et al[25] Tummaruk and Sang-Gasanee[46] Guillou et al[50]
Vaginal discharge	Blood et al[2] Martin et al[6] Persson et al[7] Hermansson et al[10]	Pendl et al[25] Tummaruk and Sang-Gasanee[46]
External signs of mastitis*	Blood et al[2] Martin et al[6] Hermansson et al[10] Ross et al[15]	Pendl et al[25] Preissler et al[44] Tummaruk and Sang-Gasanee[46]
Hungry piglets	Blood et al[2] Ross et al[15]	Pendl et al[25] Preissler et al[44] Claeyé et al[45] Guillou et al[50]

* Clinical signs absent in the subclinical variant.

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Conflict of interest

None reported.

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